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# Pathology of Right Ventricular Failure Following LVAD Implantation

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# Pathology of Right Ventricular Failure Following LVAD Implantation

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The use of left ventricular assist devices (LVAD) in heart failure patients as bridge to transplant or as stand-alone therapy is becoming increasingly common. However, many LVAD recipients unpredictably develop *de novo* right ventricular (RV) failure, a leading cause of mortality in this population. Consequently, it is crucial to understand the pathology of RV failure in order to better understand this phenomenon and develop treatments for such patients. The goal of this project is to define the pathology of post-LVAD RV failure. To accomplish this we compared clinical data and tissue samples obtained from four groups of patient: normal RV function on medical or inotropic therapy (control); RV failure on medical or inotropic failure (chronic RV failure); post-LVAD with normal RV function; and post-LVAD with RV failure. Using these four groups, we analyzed interstitial and replacement fibrosis, cardiomyocyte hypertrophy, capillary density, and gene expression and ontology. Through this analysis, we demonstrated that the pathology of post-LVAD RV failure represents a distinct entity from chronic RV failure. Patients with post-LVAD RV failure displayed increased replacement fibrosis, decreased cardiomyocyte hypertrophy, and increased capillary density. Surprisingly, patients with apparent normal RV function post-LVAD displayed a similar pathology, suggesting that disease process may affect more patients than previously anticipated. Finally, RNA sequencing revealed alterations in pathways associated with RNA splicing, protein catabolism, and intracellular localization. Collectively, these observations provide pathological and molecular evidence that post-LVAD RV failure represents a distinct disease entity and highlights the importance of an improved understanding of LVAD associated complications.